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PARASITISM AND A SHORTAGE OF REFUGES JOINTLY MEDIATE THE STRENGTH OF DENSITY DEPENDENCE IN A REEF FISH

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Abstract. Various predator–prey, host–pathogen, and competitive interactions can combine to cause density dependence in population growth. Despite this possibility, most empirical tests for density-dependent interactions have focused on single mechanisms. Here we tested the hypothesis that two mechanisms of density dependence, parasitism and a shortage of refuges, jointly influence the strength of density-dependent mortality. We used mark–recapture analysis to estimate mortality of the host species, the bridled goby (*Coryphopterus glaucofraenum*). Sixty-three marked gobies were infected with a copepod gill parasite (*Pharodes tortugensis*), and 188 were uninfected. We used the spatial scale at which gobies were clustered naturally (~4 m^2) as an ecologically relevant neighborhood and measured goby density and the availability of refuges from predators within each goby's neighborhood. Goby survival generally declined with increasing density, and this decline was steeper for gobies with access to few refuges than for gobies in neighborhoods where refuges were common. The negative effects of high density and refuge shortage were also more severe for parasitized gobies than for gobies free of parasites. This parasite has characteristics typical of emerging diseases and appears to have altered the strength of a preexisting density-dependent interaction.

Key words: *bridled goby; competition; coral reefs;* Coryphopterus glaucofraenum; *density dependence; emerging diseases; fishes; mark–recapture; mortality; parasites; predation; refuges.*

REPORTS

INTRODUCTION

The stability of populations and their response to environmental perturbations is shaped by the strength and nature of density-dependent feedbacks in population growth (Murdoch 1994). Density dependence in population growth can have a variety of underlying biological causes, including various types of predator–prey, host–pathogen, and competitive interactions (Lomnicki 1988). Isolating these interactions and defining their effects on natural populations remains a major challenge in population ecology (Turchin 1999, Krebs 2002). One source of difficulty arises from the possibility that multiple density-dependent interactions might operate simultaneously (May 1983, den Boer and Reddingius 1996, Polis et al. 1998, Krebs 2002). Despite this possibility, for practical reasons, most attempts to identify and quantify the influence of density-dependent interactions have focused on single mechanisms (Holmes 1995).

Here we focus on the possibility that two mechanisms of density dependence, parasitism and competition for refuges, might combine to influence the strength of density-dependent mortality. Either of these mechanisms in isolation has the potential to induce density dependence, though there are relatively few experimental field studies that provide support for either hypothesis. Many animals use structural features of their habitat to take refuge when threatened or attacked by predators. When the number of such shelters is limited, mortality inflicted by predators is likely to increase as prey density increases (Murdoch and Oaten 1975). Competition for a limited supply of refuges is thus a potentially widespread cause of density-dependent mortality, and cross-factored manipulations of refuge availability and density provide direct support for this hypothesis (e.g., Seitz et al. 2001, Holbrook and Schmitt 2002, Forrester and Steele 2004 and references therein).

There is also a handful of studies showing that parasites can inflict density-dependent mortality and/or fecundity in nature (Tompkins and Begon 1999) and, in two of these cases, this density dependence is sufficient to regulate wild-host populations (Hudson et al. 1998, Albon et al. 2002). The paucity of evidence for parasite-induced density dependence is partly because experimentally infecting or disinfecting populations to properly isolate the effect of parasitism is difficult in nature (Tompkins and Begon 1999). Identifying infected hosts nondestructively and tracking their fates is also difficult in many systems (Plowright 1988).

Interactive effects of parasitism and a shortage of refuges that mediate the strength of density dependence are plausible (Hassell 1982), especially since parasite-induced changes in host behavior can affect a host's susceptibility to predators and/or its competitive ability (Barber et al. 2000). We used a study species for which

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TABLE 1. Census details and release dates of marked gobies.

Census	Census date	No. gobies censused	Parasite prevalence (%)	No. gobies marked	
				Unparasitized	Parasitized
1	28 Aug–1 Sep	1823	8.9	70	16
2	9–12 Sep	1565	10.4	60	18
3	22–25 Sep	1166	7.8	54	18
4	7–9 Oct	964	13.2	4	11
5	14–17 Oct	834	13.9	0	0
6	21–23 Oct	852	8.9	0	0

Note: The study was conducted at a shallow site on the wave-protected south side of Guana Island, British Virgin Islands.

field experiments show that individuals have progressively diminishing access to refuges as they become crowded. Crowded individuals are more vulnerable to predation than those at lower density, which leads to density-dependent mortality (Forrester and Steele 2000, 2004). Mortality of this species is also increased by infection with a common macroparasite (Finley and Forrester 2003). This system thus provided the opportunity to test the hypothesis that parasitism increases the strength of density-dependent mortality by exacerbating the effects of refuge shortage.

METHODS

The study system

The host species, the bridled goby (*Coryphopterus glaucofraenum* Gill), is a small, benthic, reef fish that is common throughout the Caribbean. Like most reef fishes, bridled gobies produce pelagic larvae. Larvae settle to reef habitats at 6.5–8 mm standard length (SL) after roughly 30 d in the plankton. Juveniles mature into females at 22–25 mm SL and can change sex to become males at ~30 mm SL (Cole and Shapiro 1992). The largest individuals reach 50–55 mm SL, and very few live beyond a year. Bridled gobies occupy small, stable home ranges (~2 m^2) in areas of interspersed sand and reef. A mix of sand and reef is required because they feed on sand-dwelling invertebrates, but use crevices at the reef/sand interface as temporary refuges when threatened or attacked by predators (Forrester and Steele 2004).

The pathogen is a parasitic copepod (*Pharodes tortugensis* Wilson), which commonly infects the gill cavity of bridled gobies. This copepod is reported to infect other fish species (Ho 1971), but at our study site was only found infecting bridled gobies and two ecologically similar congeners, *C. dicrus* and *C. eidolon* Böhlke & Robins (Petrik-Finley 2005). Parasites were never found on newly settled gobies, suggesting that gobies become infected only after settling to the reef. The parasite has a direct life cycle and can infect new hosts soon after hatching. Laboratory studies show that parasites are transmitted among neighboring gobies, but the rate of transmission is quite low, and a correlation between host density and transmission was not detectable statistically (Petrik-Finley 2005). Infection intensity increases with fish size, and a typical infection consists of several smaller male and juvenile copepods and 1–2 larger females. Most infected fish (65%) were intermediate in size, 15–30 mm SL, and very few fish >35 mm SL were infected (Petrik-Finley 2005).

This study was done at a shallow (5–7 m) site on the wave-protected south side of Guana Island, British Virgin Islands (18°29′ N, 64°35′ W). The site comprised a rectangular area of mixed sand and reef (30 × 26 m) embedded within a larger expanse of similar habitat. To facilitate censusing gobies and mapping habitat, we placed markers to subdivide the site into a 2 × 2 m lattice.

Mark–recapture protocols

A sample of gobies resident within the site was marked in situ and released on each of four dates in 2003 (Table 1). Gobies were captured using hand nets and anesthetic (Quinaldine; Sigma-Aldrich, St. Louis, Missouri, USA) and measured using calipers. Fish <25 mm SL were marked by subcutaneous injection of a small spot of colored elastomer, whereas larger gobies were injected with a coded plastic tag (1 × 2.5 mm; Malone et al. 1999). Gobies were released back into their home ranges within 5 min of capture. All marked gobies were recognizable as individuals, and divers could view the marks without recapturing the fish. Gobies for marking were selected at random, with restrictions based on the fact that only gobies >15 mm SL can be marked without side effects and on the need to obtain a sample representative of the size range commonly infected with parasites. We also ensured that gobies were sampled from all parts of the site and that we obtained an adequate sample of parasitized fish. We marked 63 gobies with parasites and 188 without, ranging in initial size from 14.5 to 38.4 mm SL (mean = 22.2 mm SL).

Defining the density and refuge availability experienced by each marked fish

We censused the entire goby population at the site on six occasions spread over 56 d in 2003 (Table 1). Each census took 3–4 d to complete. Using the lattice markers as a guide, the location of each goby was recorded on a map of the site and later assigned a Cartesian coordinate (x, y). We also made a visual diagnosis of the presence or

absence of parasites. Visual diagnosis of infection status was based on the fact that infected gobies have a distended operculum. Previously, we captured and dissected fish after making these visual diagnoses, which showed that 93% of uninfected fish were correctly diagnosed (111 of 120 correct), whereas diagnosis of infected fish was 85% accurate (57 of 67 correct). Most errors in diagnosis occurred because fish infected with a few small copepods are hard to distinguish from parasite-free fish (Petrik-Finley 2005).

In a companion study at our study site, spatial statistical models (Ripley's K) revealed strong clustering of gobies in patches ~4 m^2 (Petrik-Finley 2005). We therefore used a "neighborhood" of this size to characterize the density experienced by each marked fish. For each marked goby, we calculated the number of conspecifics within a 2 × 2 m area centered on the midpoint of its home range. Densities were calculated for each date on which the goby was alive, and we used the time-averaged density when testing for density-dependent mortality.

Crevices suitable for use as refuges by gobies are usually at the junction between sand patches ($\geq$0.4 m^2 in area) and rocks (>5 cm diameter) or corals. To estimate the density of refuges available to each marked goby, we mapped habitat within the site on 13 and 18 October 2003. Divers hovered over each 2 × 2 m subsection of the site and sketched the outline of all rocks (>5 cm diameter) and corals. A grid of 250 regularly arrayed points was overlain on each sketch and the type of substratum under each point noted. We again centered a 2 × 2 m window over the midpoint of each goby's home range and used the array of points to calculate the percent cover of rock/coral within the neighborhood of each marked goby. Previous surveys revealed a hump-shaped relationship between refuge density (y) and the percent cover of rock/coral (x), which peaks at ~40% cover (G. E. Forrester, *unpublished data*). We thus used piecewise linear regressions to predict refuge density (number per square meter) within the neighborhood of each marked goby (for 0–40% cover, $y = 0.72 + 0.14x$, $n = 280$, $r^2 = 0.59$; for >40–80% cover, $y = 11.6 - 0.12x$, $n = 22$, $r^2 = 0.37$; for >80% cover, $y = 0$, $n = 6$).

Modeling survival and resighting probabilities

We used the resighting histories of marked individuals to model survival (ϕ) and resighting probabilities (p) (Lebreton et al. 1992). Individual gobies constitute replicates in all models. Our primary goal was to model the effects of goby density (density), the density of refuges (refuges), and infection status (parasitism) on survival. We also examined the effect of body size (size) because, although not of specific interest, survival is often lower in small fish than larger ones. We used the logit link function to incorporate these predictor variables into models and used the small sample version of Akaike's information criterion (AIC_c) and evidence ratios (w_i/w_j) calculated from Akaike weights (w) to evaluate model fit (Burnham and Anderson 2002).

Evaluating the effect of parameters of interest on survival from mark–recapture data requires first identifying an acceptable starting model (notation $\phi(x)p(y)$) and then controlling any biases in resighting probability. Since parasitism is a binary variable and we know that parasites affect goby survival (Finley and Forrester 2003), we constructed all models separately for fish with and without parasites. Our data failed to meet the assumption of the widely used Cormack-Jolly-Seber (CJS) starting model, which allows ϕ and p to vary relative to parasitism and time (t). We therefore developed an alternate starting model that adequately described the data (for details see the Appendix). The alternate starting model grouped time intervals into two categories based on time since marking (TSM): one for the interval after the fish was marked and another for all subsequent intervals (ϕ[parasitism × TSM]p[parasitism × TSM]). Analysis of bias in resighting probability revealed that p was affected by goby density, so we used the model ϕ(parasitism × TSM)p(parasitism × TSM + density) as a starting point from which to test effects of the covariates on survival (for details of the analysis of bias in p, see the Appendix).

Our starting model for ϕ, expressed in the form of a linear model, is $\text{Logit}(\phi) = \beta_0 + \beta_1(\text{TSM}) + \beta_2(\text{parasitism}) + \beta_3(\text{parasitism} \times \text{TSM})$. We know from our previous work that parasitism, density, and refuges all affect goby survival and that effects of density and refuges interact (Finley and Forrester 2003, Forrester and Steele 2004). In this study, we are primarily interested in whether there are additional interactive effects involving parasitism. Therefore, all our candidate models include effects of parasitism, density, refuges, and density × refuges. We then considered progressively more complex models by adding two-way interactions (parasitism × density and parasitism × refuges), first singly, then both together. Lastly we added the three-way interaction (parasitism × density × refuges; Table 2). We hypothesized that there would be a three-way interaction because the strength of density-dependent survival would be increased where refuges were less dense, and this increase would be more pronounced for parasitized fish than for fish lacking parasites. Finally, we also evaluated models containing effects of body size, but found that survival could be adequately modeled without including its effect (for details of the analysis of body size on survival see the Appendix).

Results

Of our candidate models describing effects of parasitism, density, and refuges on survival, the model with most support in the data was the most complex model (model 1), which contained a term for the three-way interaction between these factors (Table 2). Based on the evidence ratios, this model had 3.2 times more support than the next most likely model (Table 2). The second

TABLE 2. Candidate mark–recapture models screened for effects of infection status (parasitism), goby density (density), refuge availability (refuges), and time since marking (TSM) on survival (ϕ).

Model		AIC_c	ΔAIC_c	w_i	np
1	ϕ(parasitism + TSM + [parasitism × TSM] + density + refuges + [density × refuges] + [parasitism × density] + [parasitism × refuges] + [parasitism × density × refuges])	933.6	0	0.713	14
2	ϕ(parasitism + TSM + [parasitism × TSM] + density + refuges + [density × refuges] + [parasitism × density] + [parasitism × refuges])	935.9	2.33	0.222	13
3	ϕ(parasitism + TSM + [parasitism × TSM] + density + refuges + [density × refuges] + [parasitism × density])	938.7	5.14	0.055	12
4	ϕ(parasitism + TSM + [parasitism × TSM] + density + refuges + [density × refuges] + [parasitism × refuges])	943.9	10.28	0.004	12
5	ϕ(parasitism + TSM + [parasitism × TSM] + density + refuges + [density × refuges])	943.4	9.83	0.005	11
6	ϕ(parasitism + TSM + [parasitism × TSM] + density + refuges)	947.7	14.15	0.0006	10
7	ϕ(parasitism + TSM + [parasitism × TSM])	958.4	24.82	<0.0001	8

Notes: ΔAIC_c is the difference in the corrected Akaike Information Criterion (AIC_c) between the lowest AIC_c and AIC_c of model i; the Akaike weight (w_i) is the weight of evidence in favor of model i; np is the number of parameters. For all models, resighting probability is modeled as p([parasitism × TSM] + density).

most likely model was model 2, which included both of the two-way interactions involving parasitism (Table 2). Model 2, in turn, had at least 4.1 times as much support as any of the simpler models considered (Table 2). Although there is perhaps not strong enough evidence to strongly favor model 1 over model 2, it seems safe to conclude that these two models are far better supported than any of the simpler models. Our main finding is thus that the effect of parasitism on survival is conditional both on goby density and on refuge availability.

To visualize the biologically interesting two- and three-way interactions revealed by the modeling of ϕ, we reconstituted parameter values from the final model. Plots of the parameter values indicate that the effect of parasitism is reduced when refuges are more common (Fig. 1a). The survival of gobies generally declines as their density increases, and the strength of density dependence is generally greater for fish with parasites than without (Fig. 1b) and greater in areas with fewer refuges (not shown). The three-way interaction appears to arise, however, because the increased strength of density-dependent survival in areas with fewer refuges is much more pronounced for parasitized fish than for fish lacking parasites (Fig. 1c, d).

DISCUSSION

Although previous experimental work confirms a causal link between the effects of refuge availability and goby density on survival (Forrester and Steele 2004), we have no experimental evidence to indicate a causal relationship between parasitism and mortality. Because we tracked the fate of gobies with and without parasites, both in this study and in previous studies of *P. tortugensis* impacts (Finley and Forrester 2003, Petrik-Finley 2005), we can be sure that an effect *associated* with parasitism influences goby mortality. However, since both studies are correlational, the possibility of a spurious association between parasitism and mortality remains. Keeping this caveat in mind, our results suggest that parasitism increases the strength of density-dependent mortality by exacerbating the effect of refuge shortage.

Our results do not, however, tell us precisely how the presence of parasites alters the gobies' interactions with predators and with refuges. The two main possibilities are that parasitized fish are poorer competitors for access to refuges or are simply targeted more effectively by predators than their parasite-free neighbors. There are a variety of behavioral changes induced by parasites that might lead to parasitized fish becoming poor competitors or selectively consumed by predators (Barber et al. 2000). Most predation on gobies is inflicted by larger fishes (Forrester and Steele 2000), and copepods carried by the goby are unlikely to be transmitted to a predatory fish that consumes a goby. Predation is not, therefore, a means by which parasites move from an intermediate to definitive host (Lafferty and Morris 1996). If predators selectively consume parasitized gobies, we suspect this is a simple consequence of parasitized gobies being easy to detect or capture (Lafferty 1992). Another possibility is that parasitized gobies are less able to secure access to refuges than the unparasitized gobies with whom their home ranges overlap. Aggression among gobies is more frequent as they become crowded (Forrester et al., *in press*), and agonistic encounters may have a role in securing access to refuges. The ratio of wins:losses in aggressive encounters ($n = 41$) is slightly lower in parasitized gobies (1.29:1) than in unparasitized gobies (1.44:1), but more samples would be required to

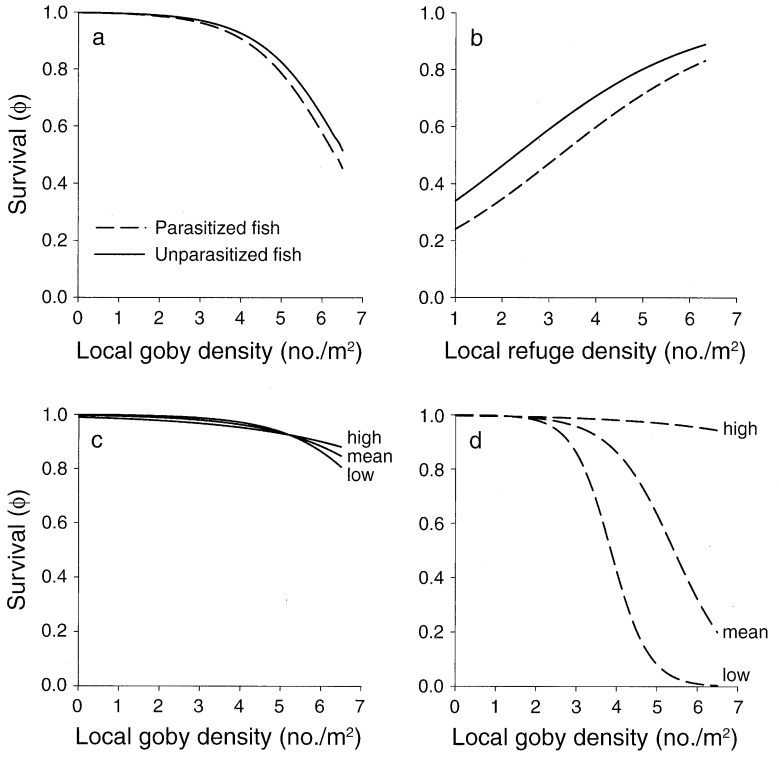

FIG. 1. Reconstituted parameter values from the final mark–recapture model to illustrate the interactive effects of parasitism, goby density, and refuge density on survival. (a) The two-way interaction between parasitism and density. (b) The two-way interaction between parasitism and refuges. (c, d) The three-way interaction among parasitism, density, and refuges. Separate plots are drawn for fish experiencing the lowest (0.86), mean (3.66), and highest (6.32) refuge density (number of crevices per square meter) at the site: (c) unparasitized fish; (d) parasitized fish. The study was conducted at a shallow site on the wave-protected south side of Guana Island, British Virgin Islands.

rigorously test whether parasitized fish lose out in competition for refuges (Finley and Forrester 2003).

Recent decades have seen dramatic increases in the number of reports of diseases affecting wildlife, both in terrestrial (Daszak et al. 2000) and marine habitats (Harvell et al. 1999). Although its distribution, host range, and prevalence is little studied, *P. tortugensis* has the characteristics of an "emerging" disease in the sense described by Daszak and colleagues (Daszak et al. 2000). Around Guana Island, the prevalence of *P. tortugensis* on *C. glaucofraenum* has increased markedly from <3% in 1993–1994 to an average close to 20% from 2003 to 2005 (G. Forrester and R. Finley, *unpublished data*). In contrast, the prevalence of *P. tortugensis* has remained extremely low (<0.05%) over the past 10 years at Lee Stocking Island, Bahamas, the other site at which we have studied *C. glaucofraenum* intensively (G. Forrester and M. Steele, *unpublished data*). A shortage of refuges from predation causes density-dependent mortality at both Lee Stocking Island and Guana Island (Forrester and Steele 2004). The emergence of wildlife diseases, both on land and in the ocean, has often been associated with increases in host mortality (Harvell et al. 1999, Daszak et al. 2000), and our previous work at Guana Island revealed that copepod parasitism was associated with a doubling of the instantaneous mortality rate in bridled gobies (Finley and Forrester 2003). This study shows, however, that the impact of parasitism depends strongly on the local density of gobies and on the local availability of refuges. The increased prevalence of this copepod parasite at Guana Island thus appears to have intensified the effect of a preexisting density-dependent interaction. Parasite prevalence is currently high enough around Guana Island (~20%) that this intensification of density dependence could increase the overall regulation of the goby population.

Some workers have argued that we should expect density-dependent interactions to be complex, with multiple underlying causes that vary in the strength of their effects in space and time (Holmes 1995). We are, however, aware of few documented examples (Hansen et al. 1999, Rodenhouse et al. 2003), only one of which involves parasitism. In that case, a severe die-off of Soay sheep was apparently due to an interaction between effects of a parasite and a shortage food (Gulland 1992). To the best of our knowledge, the specific effect of parasitism we describe in this study has not been previously documented. In light of the continuing increase in disease outbreaks in the ocean (Ward and

Lafferty 2004), it is important to further document the effects of emerging diseases on species interactions.

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APPENDIX

A description of the initial modeling of survival and resighting probabilities (*Ecological Archives* E087-063-A1).